and vomiting were frequent and the patient often vomited food eaten a day previously. With the attacks of pain there was often a sensation of movements passing over the abdomen. On physical examination the abdomen was found distended and peristaltic movements were observed on one occasion. A roentgen-ray examination revealed a perfectly patent gastro-enterostomy opening, together with a marked pylorospasm but no filling defect. The diagnosis of pylorospasm with possible ulcer was made. At operation there was no evidence of ulceration. The pylorus was found somewhat spastic, however, and a pyloroplasty was performed; the patient made an uneventful recovery and has had no further return of his former disturbance.

CASE XI.—Pylorospasm associated with a chronic gastritis in a man, aged forty-eight years, simulating carcinoma.

E. J. S. had been complaining of nausea and vomiting for the past six months. There was a loss of weight of ten pounds, poor appetite, flatulence and some abdominal pain. On physical examination no masses could be detected and there was no abdominal tenderness. The gastric secretion showed a true achylia and there was blood in the stool. The Wassermann reaction was negative and there was a definite defect at the pylorus revealed by the roentgen ray. The question of malignancy could not be ruled out. An exploratory operation was advised at which a chronic gastritis, together with a marked pylorospasm, was revealed. A pyloroplasty was performed, following which entire relief was afforded.

Summary. 1. Pylorospasm is a complex nervous phenomenon the exact etiology of which has not been satisfactorily established. Experimentally the fact has been definitely demonstrated that pylorospasm may be produced in rabbits by stimulation of the vagus and inhibited by stimulation of the splanchnics. That there is a definite association between this condition and the endocrine system is indicated by the fact that the spasm may be brought about by injection of certain extracts of the thyroids and parathyroids and inhibited by injection of an extract of adrenals. On the other hand there is but little question that changes in tonus or peristalsis are of great importance in the production of this phenomenon.

2. Pylorospasm may exist in one of three types: the neurotic, the irritative and the reflex forms. In the largest percentage of cases this condition is secondary to some irritative lesion in the stomach or is reflex from disease of some other organ. Many of these cases are promptly and completely relieved by removal of the cause, chronic appendix, gall-stones, etc. There exists, however, a purely neurotic form without any demonstrable lesion.

3. The condition can usually be recognized clinically by a careful

study of the case. In the majority of the cases slight if any pathological changes can be demonstrated about the pylorus at operation, as the general anesthetic usually completely relaxes the pyloric spasm. In advanced cases one may observe varying grades of hypertrophy in the pylorus and the pyloric antrum of the stomach.

4. The symptoms of pylorospasm are rather characteristic. These consist of pains of the hunger type appearing two or three hours after meals, which are relieved by emptying the stomach of its contents as well as by the ingestion of food; of contractions of the stomach leading to tumor formation, which disappear as the spasm relaxes; of symptoms of intermittent stagnation and hyperacidity.

5. The greatest aid afforded in the recognition of pylorospasm is by roentgenology, by means of which the nervous as well as the organic forms may usually be differentiated.

6. In the treatment of this affection medical measures should always be given a careful trial; if it be secondary to other abdominal conditions the primary disorder should, as far as possible, be overcome before treatment is directed toward the spasm. The treatment consists of overcoming the primary neurasthenia. This is most satisfactorily accomplished by means of dietetic and hygienic measures. During an attack the best results are obtained by means of hypodermic injections of morphin with atropin following a thorough lavage of the stomach. The drug most useful in the treatment of this affection is atropin given in full doses. When medical measures fail to give the desired relief, pyloroplasty theoretically is the operation of choice and practically gives most satisfactory results.

THE STRIATAL AND THALAMIC TYPES OF ENCEPHALITIS

A CONSIDERATION OF THE SYMPTOMS AND SYNDROMES REFERABLE TO THE BASAL GANGLIA IN EPIDEMIC ENCEPHALITIS.

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Epidemic encephalitis has given rise to a number of rare and interesting symptoms which are largely due to the peculiar nature of the virus and its localization in parts of the nervous system not usually involved. It is not unlikely that the toxic agent possesses a special affinity for certain cellular neurons, thus producing some of the striking clinical results which have been noted.

Of these clinical manifestations those related to the corpora striata have aroused, perhaps, the greatest interest. This is partly due to the recent advances in our knowledge of the function of these structures but also to the extreme rarity of this localization in other types of inflammatory disease. No other acute affection of the central nervous system has yielded so many and such striking evidences of involvement of the great basal ganglia as encephalitis lethargica. This has not only added materially to our knowledge of the symptomatology of these structures but has also enabled us to test the validity of our present theories concerning the functions of this important region of the brain.

While the optic thalamus is frequently involved in encephalitis the thalamic symptoms are milder and less serious than are those of the striatum, and the chief emphasis of this study will be placed upon the latter. Before taking up the question of symptomatology I will review briefly the conception of the function of these structures which the writer formulated some years ago, based on clinical and pathological studies of chorea and paralysis agitans.[1]

Fig. 1.—Vertical section through corpus striatum in paralysis agitans (primary atrophy of the pallidal system), showing *A*, caudate nucleus; *B*, internal capsule; *C*, putamen; *D*, globus pallidus. Note diminution of fiber network of globus pallidus. (Weigert-Pal stain.)

Anatomical Considerations.—The corpus striatum is a large ganglionic structure which is divided by the anterior limb of the internal capsule into two parts, the nucleus lenticularis and the nucleus caudatus (Fig. 1). The nucleus lenticularis is still further subdivided into an external segment, the putamen and an internal which is termed the globus pallidus. These anatomical divisions are based upon the gross appearance of the ganglion and are purely topographical.

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[1] Hunt, Ramsay: Progressive Atrophy of the Globus Pallidus, a Contribution, to the Functions of the Corpus Striatum. Brain, 1917, xl, p. 58.

The putamen and caudate nucleus are identical in histological structure and constitute the *neostriatum,* and while described as separate ganglia in man they have the same origin and histological characteristics, the caudate being simply separated from the putamen by the passage of capsular fibers.

The globus pallidus is composed of two segments, an inner and an outer, which is produced by the fusion of nerve fibers in the lateral and mesial medullary laminæ. This portion of the striatum is older phylogenetically and termed the *paleostriatum.* The corpus striatum may therefore be divided into two portions, a neostriatum (caudate nucleus and putamen) and a paleostriatum (globus pallidus), which differ in histological characteristics, phylogeny and, as we shall see later, in their function.

FIG. 2.—Normal cell types of the neostriatum. *A*, large motor cells of pallidal type; *B*, small ganglia cells of neostriatal type. (Nissl stain.)

Cell Types of the Corpus Striatum.—In the opinion of the writer a much greater importance is to be attached to the cellular types of this region than to the gross anatomical appearance and subdivisions (Fig. 2).

The paleostriatum (pallidum) contains aggregations of large multipolar cells which are histologically of the motor type (paleostriatal or pallidal cells). In addition to a characteristic arrangement of the Nissl granules many of these cells contain deposits of yellow pigment and resemble the motor cells of the Rolandic area and the multipolar cells of the anterior horns of the spinal cord.

The neostriatum (caudate and putamen) is composed of two cell types. Of these the more numerous are small, polygonal cells which give the characteristic histological picture of this region (neostriatal cells). Scattered among these small cells are cells of much larger size. These are giant multipolar cells, containing Nissl granules and deposits of yellow pigment, and are of the same type as the motor cells of the pallidum (giant pallidal cells of the neostriatum).

The motor or pallidal type of cell is present therefore in both paleostriatum and neostriatum while the small neostriatal cells are peculiar to the neostriatum.

The motor cells (*pallidal cells*) have long axis-cylinder processes and correspond to Type I of Golgi's classification. These pass by way of the ansa lenticularis and ansa peduncularis to important nuclei in the thalamic and hypothalamic regions and form the efferent motor system of the corpus striatum (*pallidal* or *paleostriatal system*).

The small ganglion cells of the neostriatum have short axis-cylinder processes and correspond to Golgi's Type II. They terminate in the outer or inner segments of the globus pallidus. The small neostriatal cells represent a short interstriatal system which unites the caudate nucleus and, putamen with the cells of the pallidal system (*striopallidal* or *neostriatal system*).

It is well to emphasize the fact that the neostriatum also contain giant motor cells which are a part of the pallidal system. This admixture of cells types, paleostriatal and neostriatal, is responsible for certain variations and peculiarities of the clinical picture arising from lesions in the neostriatum, which will be referred to later.

Pathological Physiology.—Our knowledge of the functions of the corpus striatum has been greatly extended by the pathological study of three chronic disorders of the central nervous system: the double athetôsis, progressive lenticular degeneration and juvenile paralysis agitans. Madame Vogt,[2] in 1911, showed that the affection called athetôse doublé, and characterized by choreo-athetoid movements, spastic diplegia (without pyramidal tract involvement) and dysarthria, was dependent upon a dystrophic lesion (état Marbré), which was limited to the structures of the neostriatum. A more recent study by Thomalla[3] has also shown that dystonia musculorum deformans, a clinical type which is closely allied to double athetosis, is also caused by a lesion of the neostriatum (putamen). It would appear from these studies that spontaneous movements of a choreiform and athetoid type associated with muscular rigidity are related to the neostriatum.

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[2] Syndrome du Corps Strié, Jour. f. Psych. u. Neur., 1912, xviii, p. 479.

[3] Ein Fall von Torsions Spasmus mit Sektionsbefund und seine Beziehungen zur athetôse doublé, Wilsonschen Krankheit und Pseudo-Sklerose, Ztschr. f. d. g. Neur. and Psych., 1918, Bd. xli, p. 326.

In 1912 Kinnier Wilson[4] described the progressive lenticular degeneration, an affection characterized by rigidity and tremor of the paralysis agitans type associated with a tendency to tonic and clonic spasms. This condition was caused by symmetrical bilateral degeneration of the lenticular nuclei, including both the putamen and globus pallidus (neo- and paleostriatum), and was associated with a lobular cirrhosis of the liver. In some cases of this disease spontaneous movements of choreiform and athetoid character were also observed and this feature was emphasized by the earlier writers, and especially by Gowers, who called the affection tetanoid chorea.

Wilson's observation showed that a more massive lesion of the striatum, involving both the neostriatum and paleostriatum, produced symptoms of paralysis agitans (muscular rigidity and tremor) associated with evidences of spontaneous movement (clonic and tonic spasm, choreiform and athetoid movements).

It was therefore clear from the syndromes of Vogt and Wilson that lesions strictly limited to the corpus striatum could be the cause of rigidity and tremor of the paralysis agitans type, tonic and clonic spasms, choreiform and athetoid movements.

In 1917 the writer was able to demonstrate in juvenile paralysis agitans widespread atrophy and loss of the large pallidal cells of the corpus striatum, and referred this disorder to a primary atrophy of this motor projection system of the corpus striatum (*primary atrophy* of the *pallidal system*). The peculiar and characteristic feature of this lesion was its limitation to a special system of motor neurons.

When the pallidal system is diseased there is produced the motor syndrome which we associate with paralysis agitans. This is characterized by paralysis of automatic associated movements, muscular rigidity and rhythmical tremor (pallidal or paleostriatal syndrome).

The writer also pointed out that the small ganglion cells of the neostriatum which were well preserved in juvenile paralysis agitans (primary atrophy of the pallidal system) showed extensive degeneration and atrophy in Huntington's chorea. The small neostriatal cells were supposed to be coördinating and inhibitory in character. Because of this elective atrophy of special systems of cells the syndrome chorea was referred to a loss of the striopallidal or neostriatal system, and the syndrome paralysis agitans was referred to a loss of the pallidal or paleostriatal system.[5] [6]

Two fundamental syndromes of the corpus striatum were therefore recognized, based upon the differences of function of these two cellular systems.

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[4] Progressive Lenticular Degeneration, Brain, 1911–12, xxxv, p. 296.

[5] Hunt, Ramsay: The Efferent Pallidal System of the Corpus Striatum: A Consideration of its Functions and Symptomatology, Jour. Nerv. and Ment. Dis., 1917, xlvi, p. 22.

[6] Hunt, Ramsay: Primary Atrophy of the Pallidal System: A contribution to the Nature and Pathology of Paralysis Agitans, Arch. Int. Med., 1918, xxii, p. 647.

1. A paleostriatal syndrome (*pallidal syndrome*) characterized by paralysis of automatic associated movements, muscular rigidity and tremor caused by a lesion of the efferent pallidal system of the corpus striatum.

2. A neostriatal syndrome characterized by spontaneous movements of the automatic associated or choreiform type caused by a lesion of the striopallidal system (neostriatal system).

This conception of the functions of the corpus striatum is not at variance with other syndromes which have been ascribed to this region but rather tends to reconcile and explain certain contradictory features. The Vogt syndrome, based upon a destructive lesion of the neostriatum, is characterized by choreic and athetoid movements spastic diplegia (without pyramidal involvement) and dysarthria. In this syndrome both cell types of the neostriatum, the small neostriatal cells and giant pallidal cells, are the seat of a destructive lesion, the clinical result of which is an admixture of the symptomatology referable to both systems. There is muscular weakness and rigidity associated with choreiform and athetoid movements. The choreo-athetosis I regard as a choreiform manifestation to which has been added a certain degree of muscular rigidity which would ensue from involvement of the motor cells of the neostriatum.

The Wilson syndrome, on the other hand, based on the progressive lenticular degeneration, produces a more extensive lesion of the corpus striatum. Here the paleostriatum and the neostriatum are both involved, the symptoms of which are paralysis, muscular rigidity and tremor of the paralysis agitans type associated with clonic and tonic spasms, and in some cases involuntary movements of a choreiform and athetoid nature.

Here again there are evidences of involvement of the two essential neuronal systems of the striatum. The paralysis, rigidity and tremor are dependent upon involvement of the paleostriatal mechanism (pallidal system) while the involuntary motor disturbances, viz.: the clonic and tonic spasms and choreo-athetoid movements, would be occasioned by a destruction of the striopallidal system (neostriatal mechanism).

Involvement of the pallidal system would cause a paralysis of movements of the automatic associated type, while involvement of the striopallidal system releases this motor center from control, with the development of spontaneous movement of the automatic associated type. The one is a *paralytic lesion* and the other a *discharging lesion* in the sense that these terms were used by Hughlings Jackson.

The great variation in the symptomatology of the corpus striatum would therefore be dependent upon the relative degree of involvement of these two systems.

Functions of the Corpus Striatum.—The corpus striatum may therefore be regarded as a higher coördinating motor center which presides over the realm of automatic and associated movements. It stands in close relationship with the important correlating sensory station, the optic thalamus, and through this structure with the peripheral sensory mechanism, the cerebellum and the cerebral cortex.

In the lower forms of life, before the appearance of the pyramidal tracts and the cerebral cortex, it is the chief coördinating motor mechanism, which by its connection with important nuclei of the hypothalamic region controls and directs the activities of the extrapyramidal motor system. Like the other lower motor centers it is under the control of the cerebral cortex, but also having important independent functions, and when destroyed there is a corresponding disturbance of motor function. This disturbance of motor function may manifest itself in various ways, depending upon the localization and degree of involvement of its two essential fiber systems.

Striatal Types of Epidemic Encephalitis.—During the recent epidemic of encephalitis lethargica all of the symptoms which we commonly associate with affections of the corpus striatum have appeared in one form or another. These manifestations have been slight or severe, transitory or progressive, general or local. In addition many bizarre and fragmentary motor manifestations have been observed which were probably also of striatal origin.

My personal experience is based on the study of twenty-five cases in which evidences of striatal involvement were present at some period of the disease. Many of these were observed in the wards of the Neurological Institute, the New York Hospital and the Montefiore Hospital for chronic invalids. Others I saw through the courtesy of Dr. Tilney, Dr. Casamajor and Dr. Kennedy at the Vanderbilt Clinic or in Bellevue Hospital.

It is not my intention to describe in detail any of these cases but simply to analyze briefly, in a general way, the symptoms of striatal involvement which the epidemic called forth and their relation to our present conception of the function of these ganglia.

Modes of Onset.—Symptoms referable to the corpus striatum occur very frequently in the acute stage of the disease and are evidently associated with an early localization of the inflammatory process in this region. They may also appear at a later period after all acute symptoms of the disease have subsided. Very remarkable is the appearance of striatal symptoms as late sequelæ, weeks or even months after apparent recovery, suggesting a recrudescence of the infectious process.

These special symptoms are accompanied or preceded by the other well-known general or focal symptoms of encephalitis lethargica which serve to give the characteristic stamp to the clinical picture and remove all doubts as to diagnosis.

Symptoms of Striatal Involvement.—Striatal symptoms are of two types corresponding to the two chief syndromes of the corpus striatum, as already defined.

There is a *paleostriatal* or *pallidal type* characterized by paralysis agitans and there is a *neostriatal type* characterized by choreiform movements.

While these two clinical syndromes may appear in pure form there is often an admixture of the two, as a result of which many curious clinical pictures are produced. The *mixed striatal* type is characterized by a combination of the symptomatology of both paralysis agitans and chorea, a *mixed paralysis agitans—choreiform type.*

Of the two the paralysis agitans type is the more frequent and usually the more severe in its manifestations. This is probably due to the course and relations of the pallidal system and the closer proximity of the paleostriatum to the midbrain, which is the chief center of the inflammatory area. Of the 25 cases which came under my personal observation 18 were of the paralysis agitans type, 4 of the choreiform type and 3 were mixed striatal types.

Paleostriatal or Pallidal Type (Paralysis Agitans Type).—The paralysis agitans type is characterized by a fairly acute involvement of the voluntary musculature. Within a brief period of two or three days there develops the typical muscular rigidity, postural deformities, masklike expression of the face and the paralytic disturbances of motility characteristic of paralysis agitans. Generally speaking the tremor is much less constant and when present less conspicuous than in true paralysis agitans. This is probably caused by the sudden development of massive rigidity which masks the tremor, producing the clinical type known as *paralysis agitans, sine tremore.*

When tremor is demonstrable at this stage it is usually slight and localized in the tongue, face or hands. The characteristic pill-rolling movement which we associate with paralysis agitans is quite rare and I have only observed it as a late residual symptom in 2 cases, and never in the acute stage. On the other hand the muscular rigidity, the paralysis of automatic associated movements, the masklike expression of face, the posture of the hands and arms, the gait and attitude appear early and are identical with those observed in true paralysis agitans.

One side is frequently more affected than the other and *hemilateral types* are encountered in a late stage of the disease. Even more limited forms occur (*segmental types*), as, for example, isolated involvement of the face (the Parkinsonian mask). Involvement may also be limited to the upper or lower extremities. The segmental types I have only observed after the subsidence of the acute stage as a late residual manifestation.

The tremor may show a similar limitation and typical rhythmical tremor may be hemilateral or sometimes segmental, *i. e.*, confined to the head, an arm or a leg. These fragmentary clinical types are usually encountered only as late or residual symptoms.

The rhythmical tremor when present is often coarse and increased by movement. It is then an action tremor and is increased not diminished during the passage of a movement, in this respect differing from the classical tremor of paralysis agitans, but is very similar to what I have observed in the earlier stages of the juvenile paralysis agitans (primary atrophy of the pallidal system). The tremor is not, however, of the intention type and diminishes as the finger tip approaches the tip of the nose. The gait and attitude, the position of the hands and arms and the facial expression differ in no respect from those observed in paralysis agitans. The difference is in the course of the disease and the nature of the pathological lesion.

True paralysis agitans is produced by a progressive lesion of the *pallidal system,* which is atrophic or degenerative in nature while the paralysis agitans of encephalitic origin is merely a symptomatic manifestation due to injury of the pallidal system by inflammatory lesions or toxins. As the acute lesions subside the clinical symptoms disappear, which explains the often rapid improvement in this group of cases. We are not accustomed to this favorable outcome in true paralysis agitans, which is a progressive disorder. Similar recoveries are, however, noted after inflammatory lesions affecting other motor systems, and it is only interesting to note that the pallidal system is no exception in this respect.

A *cataleptic type* of encephalitis has also been described. The association of general muscular rigidity of paralysis agitans type with the peculiar lethargy characteristic of the epidemic encephalitis produces a clinical picture closely resembling catalepsy (flexibilitas cerea). And while true catalepsy has been observed during the course of the epidemic, many cases of so-called *cataleptic type* probably belong to the paralysis agitans group and are of striatal origin.

Hesnard[7] has suggested the existence of a transition form bridging the gulf between the paralysis agitans and cataleptic types, in which the striatum and the cerebral cortex were both affected.

Abortive Types. It is also interesting to note that in the acute stage of the disease mild transitory striatal symptoms are sometimes present. These consist of a certain tightness or tenseness of the musculature, a certain monotony and mild fixity of expression, with slight tremors of the tongue, face or extremities, which disappear as the acute symptoms subside. Cases of this type do not, however, always abort and these initial symptoms may be the forerunners of more serious striatal involvement.

Progressive Type. A tendency to progression in the paralysis agitans group is not uncommon. In my experience this tendency has been greater in those cases in which the symptoms make their appearance late in the disease or as relapses after improvement or apparent recovery. It would appear to depend upon a renewal of the inflammatory process or an actual lighting up of old lesions.

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[7] Mental Disturbances with Epidemic Encephalitis, Encephale, Paris, 1920, xv, p. 443.

A *relapsing* form, unfortunately not uncommon, is a well-recognized type of the disease. Its existence has been emphasized by von Economo,[8] Buzzard and Greenfield.[9] I have also observed two very striking examples of the *mixed striatal type,* in which there was a well-marked tendency to relapses extending over a period of one year. Careful histological study of a relapsing case by von Economo showed the existence of both old and recent lesions, side by side, which explains the peculiar features of the clinical picture.

The *sporadic type* of striatal encephalitis. Mention has already been made of the extreme rarity with which the corpus striatum is involved in other acute inflammatory affections of the nervous system. It is especially noteworthy that epidemic poliomyelitis, which gave rise to so many different clinical types, so far as I am aware, produced no examples of striatal involvement. It is therefore interesting to quote this observation made by Nixon[10] at the Bristol Royal Infirmary a number of years ago.

A boy, aged seven years, was admitted to the infirmary with headache, lethargy and rigidity of the left arm and leg. The teeth were tightly clenched and nasal feeding was resorted to. The cerebrospinal fluid was clear and there was no increase of cells. Later a general hypertonicity and rhythmical tremor developed and the patient left the infirmary with the typical picture of paralysis agitans.

Such a case is interesting as a probable example of sporadic encephalitis localized in the striatum. In a previous paper I have already reported the occurrence of a sporadic type of epidemic myoclonus multiplex[11] (epidemic encephalitis), and it is more than likely that many of the clinical types with which we have become familiar during the epidemic made their appearance occasionally in the sporadic form.

Pallidopyramidal Types of Encephalitis. In my previous studies of the symptomatology of the efferent pallidal system of the corpus striatum[12] attention was directed to the existence of *pallidopyramidal types* of *palsy.* This form is characterized by a combination of the symptoms of both spastic paralysis (pyramidal tract system) and paralysis agitans (pallidal system), and is by no means uncommon because of the close anatomical relationship of the internal capsule and the corpus striatum. In the pallidopyramidal type the paralysis is more complete because both the pyramidal and the extrapyramidal systems are involved, the one controlling isolated synergic movements of cortical origin and the other striatal movements of the automatic associated type. The muscular hypertonicity is also greater and combines the peculiar features of spasticity and paralysis agitans rigidity. Especially characteristic of the pallidopyramidal type are exaggerated tendon reflexes, clonus and the Babinski phenomena of the spastic type in conjunction with a loss of automatic associated movements of striatal type. It is rather remarkable that in my series of 25 cases of striatal encephalitis definite involvement of the pyramidal tracts was only noted in 3 cases.

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[8] Encephalitis Lethargica Chronica, München. med. Wchnschr., 1919, lxvi, p. 1311.

[9] Lethargic Encephalitis, its Sequelæ and Morbid Anatomy, Brain, 1919, xlii, p. 314

[10] Paralysis Agitans after Encephalitis: Observed a Number of Years before the Epidemic, 1917-18, Bristol Med. and Chir. Jour., 1920, p. 25.

[11] Hunt, Ramsay: Acute Infectious Myoclonus Multiplex, Jour. Am. Med. Assn., 1920, lxxv, p. 713.

[12] Hunt, Ramsay: Primary Atrophy of the Pallidal System: A contribution to the Nature and Pathology of Paralysis Agitans, Arch. Int. Med., 1918, xxii, p. 647.

This is all the more peculiar as we are dealing with a rather diffusely disseminated inflammatory reaction, a type of lesion which one would naturally expect to involve the adjacent pyramidal system of the internal capsule and pes pedunculi. It is possible that certain toxins of encephalitis may possess a special affinity for the cellular system of the striatum which render it particularly susceptible to the infection.

The *neostriatal type* (*choreiform type*). The other essential syndrome of the corpus striatum which may develop as a result of epidemic encephalitis is chorea. The choreiform movements of striatal origin are involuntary and irregular in character and are of the automatic associated type. According to the view which I have already expressed they represent a motor discharge of the striatal mechanism in contrast to paralysis agitans which is a paralytic manifestation.

As was indicated in the preliminary sketch, this disorder of motility is ascribed to a loss of the *striopallidal* system or small ganglia cells of the *neostriatal mechanism*, which are supposed to coördinate and control the purely motor functions of the striatum. A degenerative or destructive lesion of this inhibitory system would release the purely motor mechanism from control and allow the development of the involuntary automatic-associated movements of chorea.

The *choreic movements* may be general, hemilateral or segmental (local) in their distribution. They also vary somewhat in character, *e. g.*, mild and severe, large and small amplitude, and occasionally rhythmical. A certain degree of hypertonicity is sometimes present, giving an athetoid character to the movement.

Athetosis or choreo-athetosis I would regard as an admixture of chorea and muscular rigidity due to involvement of both the neostriatal and pallidal systems.

Acute Choreiform Type. Some writers on the subject of encephalitis have described cases characterized by generalized choreiform movements appearing in the acute stage of the disease, associated with delirium and other severe psychotic symptoms. A clinical picture very similar to the *chorea insaniens* of systematic writers. Archambault[13] has reported cases of this type, as have also Oehmig[14] in Munich and Dimitz[15] in Vienna. Oehmig states that of 14 consecutive cases admitted during one month of the epidemic, 11 were of this choreiform type, which would appear to indicate a form of the virus with a special affinity for the neurones of the neostriatal mechanism. The choreiform movements are, as a rule, generalized, although one side may show greater involvement. Hemichorea has also been observed (Archambault).

This acute choreiform type is apparently rare and I encountered no cases in the New York epidemic. I did observe, however, a number of cases of the "myoclonus multiplex type" which were associated with delirium. This form of movement may resemble but must be sharply distinguished from chorea. Striatal chorea is characterized by the spontaneous discharge of movements of the automatic associated type while "myoclonus multiplex" originates in the spinal and prespinal mechanism and is characterized by contraction of individual muscles or portions of muscles with comparatively slight locomotor effect (myoclonus, myokymia or fibrillary twitching).

Choreo-athetoid Types of Movements. In addition to chorea movements of an athetoid and *choreo-athetoid* type may also occur, both in the early and late stages of the disease. Of special interest is their appearance as late sequelæ months after apparent recovery. The movements of this type like those of chorea may be general, unilateral or segmental (local) in distribution. They differ from chorea in being slower, more stereotyped and associated with hypertonicity of the affected muscles.

In the more localized forms there is a peculiar repetition of slow stereotyped movements occurring at regular intervals. These, except for the limited and rather fragmentary character of the movement, are very similar in their character to other well-known forms of choreo-athetoid movements of striatal origin.

Belonging to this group are other types of movement of somewhat larger amplitude which affect more particularly the trunk and root portions of the extremities, causing curious contortions of attitude and gait. Some of these movements bear a striking resemblance to those observed in the dystonia group of motor disorders, which, since the autopsy report of Thomalla (dystonia lenticularis) must now be definitely allied with the symptomatology of the striate body.

Rhythmical Chorea (Bradykinetic Oscillation). This disorder of motility varies somewhat from the recognized lenticular types.

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[13] Choreo-athetoid and Choreopsychotic Syndromes as Sequelæ of Epidemic Encephalitis, Arch. Neur. and Psych., 1920, iv, p. 484.

[14] Encephalitis Epidemic Choreatica, München. med. Wchnschr., 1920, lxvii, p. 660.

[15] Encephalitis Choreaformis Epidemia, Wien. klin. Wchnschr., 1920, xxxiii, 163.

It is characterized by slow rhythmical movements of an extremity, sometimes involving both the arm and leg on the same side, and occurring with great regularity, eighteen to twenty movements to the minute. With the slow rhythmical oscillation of the extremity there is a simultaneous hardening of many of the muscles of the arm or leg, showing the diffuse nature and wide distribution of the muscular contractions. This form of spontaneous movement, in my opinion, is referable to the extrapyramidal system and in all likelihood to the striatal mechanism. It differs, however, in the monotonous regularity of its synchronous rhythm and stereotyped repetition from other forms of striatal movement.

The following case is a striking example of this type: The patient, a young woman, had encephalitis lethargica in January, 1920. During the height of the disease the left side of the body developed a curious spasmodic tendency which persisted after the acute symptoms had subsided. One year later, at the time of my examination, there were present hemichoreatic movements on the whole left side of the body. The movements were rhythmical and synchronous, recurring with great regularity eighteen times to the minute. The spasm was evident in the muscles of the neck, arm scapular group, abdomen and thigh and leg upon the left side. The greatest excursion was of the arm and leg, especially the former, which produced a typical movement of salutation. In the muscles of the trunk there was some tendency for the muscles of the opposite side to participate in the contraction. At times there were slight movements of the face, synchronous with the rhythmical movements elsewhere. There was no paralysis and no evidence of pyramidal tract disease. With the exception of moderate dysarthria and some fixity of the facial expression the only residual of encephalitis was this curious choreo-athetoid disorder probably of neostriatal origin. While the spasm in this case one year after the onset had not diminished, in two other patients it had gradually diminished and finally disappeared.

Marie and Levy,[16] [17] [18] who had a large experience with striatal encephalitis have described cases of this character as Brady kinetic oscillation, and emphasize the peculiar nature of this type of movement and its lack of correspondence with any recognized clinical type. While agreeing with these writers as to the unique character of this curious spasmodic type, of which four occurred in my series, there is a strong resemblance to certain types of movement which I have occasionally observed in the dystonia musculorum deformans. I would therefore regard it as related to the neostriatum.

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[16] Syndrome Excito-moteur de l'encephalite Epidemique, Rev. Neur., 1920, xxvi, p. 515.

[17] Nouveau cas de localisation—facio—masticatrice du syndrome excito-moteur tardif de l'encephalite Epidemique, Bull. et Mem. de la Soc. Méd. des Hôp. de Paris, 1920, xxxvi, p. 661.

[18] Quinze cas de mouvements involontaires apparus a la suite d'episodes grippaux ou d'encephalite lethargique, La Méd., 1920, 1, p. 270.

Acute Chorea of Childhood (Chorea of Sydenham). Before leaving the neostriatal type it will be interesting to mention some recent work on the chorea of Sydenham. Attention has already been directed to the fact that certain well-recognized types of epidemic encephalitis have been observed in sporadic form long before the existence of the present epidemic. The question naturally arises: have other clinical types of infection of the central nervous system a similar origin and relationship?

A recent pathological study by Marie and Tretiakoff[19] of the nervous system, from a case of chorea, is very suggestive from this point of view. The case was a typical one of Sydenham's chorea occurring in a child, aged ten years, and which terminated fatally ten days after the onset of the disease. Evidences of inflammatory reaction were found throughout the brain and spinal cord, with the exception of the bulb and cerebellum. The neostriatum and cerebral cortex were particularly affected. These investigators, who have also studied the lesions of epidemic encephalitis, emphasize their great resemblance to those found in the central nervous system of Sydenham's chorea.

This observation would suggest a possible relationship between the acute infectious chorea of childhood and the choreic type of encephalitis lethargica. The occurrence of neostriatal symptoms in such a case, with evidences of diffuse involvement of the nervous system, I would explain by a special affinity of the toxin for the neostriatal system of neurons (striopallidal system).

Mixed Striatal Type (Mixed Choreiform and Paralysis Agitans Types). While the paralysis agitans and choreiform types may occur in pure form it is well to emphasize the fact that these two clinical pictures are not infrequently combined in greater or lesser degree. Indeed, among the most striking features of involvement are the many bizarre combinations which unite both the elements of chorea and paralysis agitans.

In one of my cases there developed during convalescence the typical picture of paralysis agitans which progressed slowly. Four months later there were superadded to the symptoms of paralysis agitans choreiform movements of the right foot and still later typical Brady kinetic oscillations of the whole right lower extremity, which was characterized by a slow flexion of the hip, knee and ankle. This movement was of large amplitude, stereotyped and occurred at regular intervals. It lasted for two months and then gradually disappeared. The symptoms of paralysis agitans, however, continued and grew progressively worse. Two months later there was superadded a tonic spasm and athetoid contractions of the muscles of the neck and of mastication, causing distressing dysarthria and dysphagia. This case is of the progressive type and one year after the onset of the striatal symptoms showed no tendency to abatement.

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[19] Histological Examination of the Central Nervous System in the Acute Chorea of Syndenham, Rev. Neur., 1920, xxvii, p. 428.

The explanation for such clinical combinations, I believe, is to be found in the existence of two striatal systems; a neostriatal and a paleostriatal (pallidal), yielding their respective syndromes of chorea and paralysis agitans. The one a convulsive and the other a paralytic manifestation of the corpus striatum.

Rare Myoclonic Types. Occasionally one observes, after an attack of encephalitis, stereotyped rhythmical movements of the distal portion of the extremities. In one of these cases, which presented typical symptoms of paralysis agitans, there was rhythmical flexion of all the fingers, occurring about twenty times a minute. Movements of this type are so fragmentary that it is rather difficult to localize them with certainty. The association with paralysis agitans and the stereotyped regularity of the movement are in favor of a striatal origin.

Rhythmic twitchings of the face and *jaw*, either alone or in combination, have also been observed. When combined the movements of the jaw and face may be synchronous. Such manifestations may represent disjointed fragments of striatal motility or may indicate a release of kinetic impulses in subsidiary centers of the pons varolii, which contain the motor nuclei governing the facial muscles and mastication.

In one patient of my series who presented a definite sequela of paralysis agitans there was also present a rhythmical clonic movement of the jaw associated with bilateral facial spasm. The movements of the jaw and face were synchronous and occurred with stereotyped regularity about eighteen times to the minute. In another patient the only residuals of the encephalitis were the Parkinsonian mask and a lateral clonic movement of the jaw. Marie[17] has observed similar cases.

Bilateral Synchronous Myoclonia. There is still another type of movement which has been encountered in this disorder, the classification of which is somewhat difficult in the present state of our knowledge. This is a movement of the myoclonic type, involving a large number of muscles and causing bilateral synchronous movements of the trunk and root segments of the extremities occurring in very rapid tempo, fifty or sixty to the minute.

These movements differ from those of the "myoclonus multiplex type of spinal origin" in the large number of muscles which are simulataneously involved and the extent of the locomotor effect. They are also unlike those types of movements which we associate with the striatum.

Hamill[20] has described a group of cases representative of this type, in all of which the movements were increased during sleep in contrast to the ameliorating effects of sleep on the pure striatal types. He suggests their relationship to certain subordinate motor centers in the pons or medulla which act in conjunction with the respiratory center as auxillary centers of breathing.

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[20] Encephalitis with Involuntary Movements, Arch. Neur. and Psych., 1920, iv, p. 44.

I have also observed movements of this type. In my opinion they are neither cortical nor spinal, and if they do not represent lower forms of movement directly under striatal control they are probably referable to some infrastriatal motor mechanism of the paleokinetic system of motility.

The Thalamic Type.—The *optic thalamus* is the sensory counterpart of the corpus striatum. It is the great ganglionic station where the neurons of the secondary sensory paths terminate for their final grouping and distribution.

Many of these neurons terminate in the optic thalamus itself, subserving *affective sensibility*, the greater number pass to the sensory areas of the central cortex to subserve the higher form of discriminative sensibility.

Lesions in this region may produce sensory symptoms of the following character: Spontaneous pain of intolerable intensity and of persistent character; loss of superficial and deep sensibility with anesthesia; ataxia and astereognosis. There may also be present slight hemiplegia as well as choreic and athetoid movements. The sensory loss and pain are alone of thalamic origin; the other symptoms are referable to surrounding parts.

Symptoms of thalamic origin are not infrequent in cases of epidemic encephalitis (Howe[21]). They are often associated with evidences of striatal involvement, but in my experience are neither so severe nor well defined. I have observed no case presenting the complete thalamic syndrome as outlined by Dejerine and Roussy. My experience has been limited to sensory symptoms which it seemed reasonable to assume were of thalamic origin, although one could not deny their possible relationship to the central sensory mechanism of the spinal cord and brain stem.

The most frequent symptom of thalamic involvement in encephalitis in my experience has been pain. This may be of agonizing intensity and is very resistant to all analgesic remedies. It may be generalized or localized and may persist for weeks or months. It is difficult to differentiatiate localized thalamic pains from those referable to the posterior gray matter and posterior root system of the spinal cord. In differentiation I would lay some stress on the coexistence of striatal symptoms which would appear to show inflammatory involvement of both structures at the same level. In no case have I encountered any extensive anesthesia, either superficial or deep. Tactile sensation has shown little or no involvement and the chief sensory disturbances when present have been of pain and temperature sensibility. In no case was the deep sensibility affected.

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[21] The Thalamic Syndrome in Epidemic Encephalitis, Neurol. Bull., 1919, ii, 190.

From my personal experience and that of others I am inclined to believe that the thalamic symptomatology is largely limited to intractable and persistent pain associated with mild evidences of sensory loss, especially of pain and temperature sensibility.

As the optic thalamus is the sensory counterpart of the corpus striatum, and as both ganglia are closely connected by short commissural systems, it is interesting to speculate on the possible role which irritative lesions of the thalamus may play in the production of striatal types of movements, such as chorea and athetosis.

While the facts of pathological anatomy point to the neostriatum, and especially the neostriatal system, as the seat of the essential lesion of chorea, it cannot be denied that irritative foci in the thalamus or pallidum might produce spontaneous motor manifestations of the same character as those resulting from loss of its inhibitory mechanism.

Concluding Remarks.—The results of my investigations may be summarized as follows:

The large basal ganglia, and especially the corpora striata, are frequently affected in epidemic encephalitis.

Involvement of the corpus striatum produces three clinical types or syndromes:

1. A *paleostriatal* or *pallidal syndrome—the paralysis agitans type.*
2. A *Neostriatal syndrome—the choreiform type.*
3. *A mixed striatal syndrome—the combined paralysis agitans-choreiform types.*

These three types are believed to be dependent upon the existence of two distinct cellular systems within the corpus striatum.

One, the *pallidal system,* which originates in the motor cells of the corpus striatum and links this structure with important nuclei of the hypothalamic region, and controls the various motor activities of the extrapyramidal tracts. When this system is involved the symptoms of paralysis agitans develop, *i. e.,* paralysis of automatic associated movements with hypertonicity of the muscles and rhythmical tremor.

The other, the striopallidal or *neostriatal system,* which exercises a coördinating and inhibitory influence on the purely motor functions of the corpus striatum. When this system is involved, chorea or spontaneous movements of the automatic associated type develop.

The mixed striatal types result from involvement of both systems with the production of symptoms characteristic of each.

The recognition of these two systems and fundamental syndromes of the striatum serve to explain and reconcile many peculiarities of striatal symptomatology.

The *pallidal* or *paralysis agitans type* of encephalitis lethargica may be general, hemilateral or segmental in distribution. Abortive, relapsing and progressive types are also recognized.

The *neostriatal* or *choreiform type* may also be general, hemilateral or local in distribution. There is an acute choreiform type; a choreo-athetosis and athetoid and rhythmical types.

These types may occur in pure form or in combination. Of my series of 25 cases, 18 were of the paralysis agitans type, 4 of the choreiform and 3 of the mixed striatal type.

Thalamic symptoms also occur in encephalitis lethargica.

When present they consist of severe and persistent pain, with disturbances of superficial sensibility, more especially of the pain and temperature sense. Evidences of the complete thalamic syndrome are rarely if ever encountered.

VEGETABLE PROBLEMS IN DIABETIC DIETS.

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A certain eminent specialist is quoted as saying that "diabetics must not expect a varied diet." That this is not necessarily true I hope to be able to show. The vegetables in common use are only a fraction of those that mankind has found fit for food in one country or another during the thousands of years that gardening has given pleasure to the rich and the poor. There is a vast store of experience on record in the garden literature of the world which diabetics have a special motive in searching for suggestions as to vegetables of value to them and as to methods of cooking to give added variety. Americans have not begun to know the good dishes of Europe, and India, China and other ancient lands must have accumulated culinary discoveries beyond any that we dream of. I am growing this year 81 species or kinds of plants in 255 varieties, and I have accomplished the aim of having some fresh green vegetables of my own growing every day in the year without a greenhouse. I am sure that others can do the same, and I believe that the achievement will bring them new enthusiasm and contribute to the maintenance of health of body as well as of mind.

Everyone needs a hobby, diabetics particularly, to take their mind from their hunger troubles and give them an interest in life. The culture of rare or uncommon vegetables fills their need admirably. Achievement stimulates renewed effort and it is one of the advantages of gardening that there are always new fields to conquer, new crops to test, new problems of succession cropping, how to get vegetables earlier or prolong their season into the winter by storage.

Only those who have been through it can appreciate the food problems of the diabetic patient, who has to face for probably the rest of his life an altered regimen, with strict limitations of kind and of quantity of food, old likes and habits broken up, favorite dishes forbidden, temptations to transgress constantly presented, and with the influence of family and friends often misdirected and imperfect.

The difficulties of the diabetic diet center mainly around the supply of vegetables; the provision of variety at all seasons at a reasonable price and cooked and served in a palatable manner. There is nothing lacking in American soil and climate. We can grow everything somewhere or at some season. Our people, however, have not yet become sufficiently herbivorous for their own good. Meat and potatoes are too much the reliance, flanked by corn, peas or tomatoes, to the neglect of health-giving greens or salads. This reacts against the diabetic in a double way. The onset of the disease compels a greater change in his food habits, and the narrow list of vegetables in common use in the community makes it more difficult to obtain supplies in the markets.

We are further handicapped by our primitive methods of cooking. Many cooks seem to know but one way of cooking vegetables; that is, simply boiling and serving *au naturel.* Vegetables that are young and tender and fresh from the garden require little dressing; but, nevertheless, plain boiled spinach gets horribly monotonous if taken twice daily, month in and month out, and our patient is excusable who breaks diet or gets to doubting whether life is worth living.

A wonderful change has been made in the outlook for diabetics by the discoveries of Dr. Frederick M. Allen and his co-workers. To the student of nutrition we have the most interesting disease in the list. Our chances for a useful existence have been multiplied. The treatment is exact. Our diet can be calculated to the gram and checked constantly by simple tests. Old medical traditions are broken, in that the patient is admitted to a full knowledge of the treatment and trained to handle his own case. His chances for long life may be greater than his healthy brothers.

But the problem of food supply stands where it was, and in this field there is great opportunity for progress and improvement. It is not to be solved by proprietary foods or commercial compounds. They may be useful in special cases, but reliance may better be placed upon Nature's original sources.

There is need to spread the information that many unheard-of vegetables are in existence; that they may be grown with ease, and if demanded by a number of consumers would be supplied by the market gardeners.

Diabetics may be divided into two classes: Those who possess gardens and those who do not. The latter, naturally in the majority, constitute a special problem requiring a new line of effort. Many of the vegetables which are recommended below are to be had only in the larger city markets for a very short period of the year. Many are never seen in the market. How can they be had by the non-gardeners? The physician can assist in creating the demand by